Ottow [5] records a case due to the secondary infection of a large piece of retained placenta; and Doederlein [6] a case of acute anteflexion due to an old ventrofixation occluding the cervical canal and preventing normal delivery, with secondary infection in the lacerated tissues, causing physometra; while Frank [7] states that physometra may develop if gas-producing organisms penetrate secondarily in any gynecologic or obstetric condition.

REPORT OF CASE

Mrs. ———, a widow, age thirty-six, who had always been in good health, was referred to us for pain in both inguinal regions, over the sacrum, in the bladder, which was accompanied by a rather profuse foul-smelling discharge. There had been no bleeding other than the normal menstrual amount.

The patient stated that shortly after her last period, ten days previously, she began, for the first time, to have pain low down in her sides and the discharge which had gradually increased and assumed the odor complained of. She had been in bed for the last two days with a slight temperature, she believed, as she had not summoned a physician. She has been widowed two years, and has one child six years of age whose birth history is normal. She states that her sexual and menstrual life have always been normal.

She admitted occasional sexual contact; states that she had missed no periods. She was very critically questioned as to interrupted pregnancy, but denied it definitely.

Examination at home, revealed a well-built young female apparently not very ill, lying comfortably in bed. Temperature was 99.6; pulse, 115; and with no remarkable physical findings except the following: Some slight tenderness over the pubis and in both inguinal regions, but no rigidity; the uterus just palpable at the pubis, but abdominally not tender. Her outlet showed a moderate relaxation, but no cystocele or rectocele; the urethral orifice was negative.

Vaginal examination showed some tenderness in both adnexa, but no masses. There was a slight erosion on the posterior lip of the cervix, a moderate discharge which had the combined odor of *B. coli* and putrefaction; the uterus appeared about twice its normal size to palpation.

The diagnosis of erosion, endocervicitis, and probable low-grade subacute pelvic inflammatory was made. She was placed upon expectant treatment and P. M. C. douches and told to report to the office when able.

Three days later, in the office, because of the discharge and its odor, a sterile swab was inserted into the cervical canal for the purpose of making smears. There was a slight resistance just within the external os which when overcome suddenly released a small amount of gas, foul-smelling and accompanied by a decidedly audible "hiss." After appropriate preparation a sterile probe was inserted further into the uterine canal, with a repetition of the same phenomenon. The diagnosis of physometra, as an accompaniment of multiple strictures due to a partially obliterating endocervicitis, was added.

Laboratory Report.—The following laboratory report is presented through the courtesy of the Mount Zion Hospital clinical laboratorv.

Twenty-four-hour culture of material from cervix and uterine canal: *B. coli,* four plus; *Streptococcus haemolyticus,* two plus; *Streptococcus viridans,* two plus (green pigment forming colonies); Gram-negative diplococci, two plus (*M. catarrhalis* ?).

A moderate cervical dilatation was performed in the office, and further treatment was conservative. The cervical canal was cleaned out three times weekly with hydrogen peroxid and followed with a tampon impregnated with 0.2 per cent formalin in pure glycerin, and she was instructed to use the standard P. M. C. hot douche at least three times daily. She was also given capsules of quinin, grains five, and ergotin, grains one, three times a day after meals to restore uterine tone.

Her discomfort and fever disappeared almost immediately, and she has remained free from such as well as the odor for the past two weeks. Her uterus is normal size; and although there have been no symptoms or signs of malignancy she has been advised to have a diagnostic curettage, with cauterization of the cervix.

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PERFORATED GASTRIC ULCER IN A PATIENT WITH TABES DORSALIS

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ACUTE upper abdominal pain in a patient with known tabes dorsalis, especially if accompanied by vomiting, usually is interpreted as due to a gastric crisis. Coincident occurrence of an acute abdominal surgical condition with tabes is rare, but failure to recognize such coincidence in these patients is disastrous.

We report the following case primarily because of its relative rarity, secondarily to emphasize the necessity of a rigid diagnostic scrutiny of every tabetic patient with severe abdominal pain.

REPORT OF CASE

Mr. D. B. S., age forty-two, had been diagnosed as having tabes dorsalis for ten years, with the usual findings of a sluggish pupillary light reflex, absent patellar reflexes, incoördinate gait, and a strongly positive blood Wassermann reaction. He had suffered intermittently for years with some postprandial epigastric distress, which he had interpreted as due to his blood disease. About six months previous to his present sickness he was seized with severe epigastric pain and vomiting. He was seen by two physicians, and a diagnosis made of a gastric crisis. He was relieved by a hypodermic of morphin, and in a few days was apparently as well as ever.

On October 29, 1926, he had some dull epigastric distress and took nothing but liquids. About eleven at night he suffered an acute attack of mid-epigastric pain. He did not vomit immediately, but, believing vomiting would relieve him, took some fluid extract of ipecac and vomited some "brownish liquid" in the toilet. This was not saved. He was seen at twelve midnight. At this time there was slight rigidity of the upper right rectus muscle. The temperature and pulse rate were normal. He did not appear to be in acute pain.

In view of his known tabetic condition, and the knowledge that he was supposed to have had former attacks of gastric crisis, the latter diagnosis was made tentatively.

A hypodermic of one-quarter of a grain of morphin did not relieve him, and in thirty minutes another one-quarter of a grain was given. This relieved him slightly.

At six the next morning, he was suffering more acutely, there was marked generalized abdominal

rigidity, particularly right-sided, and tenderness in the lower right quadrant. The temperature had risen to 99.4 degrees.

A diagnosis of a perforated viscus, probably a gastric ulcer, was made. He was sent to Saint Vincent's Hospital, where Dr. T. C. Myers saw him, concurred in the diagnosis, and immediately operated. A preoperative leukocyte count was 18,126, with 80 per cent polymorphonuclear cells.

An acutely perforated gastric ulcer on the lesser curvature near the pylorus was found. The opening was about three-quarters of an inch in diameter, with thin sclerotic edges. There was a large amount of gastric contents in the peritoneal cavity.

The opening was closed without posterior gastroenterostomy due to the relatively poor condition of the patient. It had been over six hours since the perforation had occurred. Postoperatively his condition was precarious for several days, but then he steadily improved, and was discharged five weeks later in good condition. Following the operation he still had sporadic trouble, with some postprandial distress and pyrosis, but was comfortable when he was faithful to a bland ulcer diet.

Four and a half years later he suddenly developed severe pain in the epigastrium and umbilical region. At this time he was in a small town seventy-five miles away, and was seen by another doctor. Two days later he was vomiting continuously, and Dr. T. C. Meyers was consulted over the telephone. The possibility of a perforated viscus was stressed and the patient advised to come in to the hospital. This he refused to do until the fourth day, April 5, 1931, after the development of the severe pain, when he was admitted to Saint Vincent's Hospital.

At the time of admission the temperature was 102.4 degrees; the pulse was only 88. There was no abdominal distention or rigidity, but peristalsis was definitely diminished and the leukocyte count was 21,000. A tentative diagnosis was made of perforated gastric ulcer, with generalized peritonitis.

On April 10, 1931, he developed signs of hypostatic congestion in both lungs. His condition became rapidly worse and he died on April 13, 1931.

Necropsy.—At necropsy the site of the ulcer found at operation in 1926 was healed, but in the first part of the pylorus, on the posterior wall, was an ulcer about 1.5 centimeter in diameter and 5 centimeters deep, firmly adherent to the pancreas and mesocolon. There was acute generalized peritonitis, with about 100 cubic centimeters of thick pus in the pelvis. The ulcer evidently had ruptured, then became sealed over by the mesocolon. The histologic examination of the ulcer showed an increase of interstitial fibrous tissue in the muscularis layer, the latter infiltrated with round cells and mononuclear cells.

COMMENT

Stokes [1] in "Modern Clinical Syphilology" describes three types of gastric crises, namely, "the attack of pain without vomiting; the attacks of vomiting without pain; and the most common type, combining both pain and vomiting. Many gradations from abortive to severe forms exist."

It rarely occurs that a perforated peptic ulcer, or another acute abdominal condition exists in the tabetic patient with symptoms of one of the above types, and the diagnosis is apt to be gastric crisis. It is none the less as feasible as the reverse, where the gastric crisis mimics other abdominal disease.

Gastric syphilis with a syphilitic gastric ulcer may occur, but is rare. The coexistence of a nonspecific gastric ulcer with tabes is not so rare. Perforation in either type is a potential menace, and an accurate diagnosis and immediate surgery in such event is essential.

Hunt and Lisa [2] recently reported four cases of tabes dorsalis with associated duodenal ulcers, in only one of which was the ulcer suspected before death. Two died from hemorrhage, one from an acute perforation, and one from peritonitis following a posterior gastro-enterostomy.

In the case here reported, the correct diagnosis was nearly missed, and then made only after the time limit usually considered safe for surgical repair of a ruptured peptic ulcer.

It is interesting to note that the rupture of the second ulcer was due to a new lesion, as the necropsy revealed healing at the site of the first ulcer. The histologic findings were those of a simple ulcer, and suggest the same etiology as that of any peptic ulcer.

CONCLUSION

The investigation of the gastro-intestinal tract of the patient with tabes dorsalis who has gastro-intestinal symptoms, to eliminate possible organic lesions would seem strongly indicated in the light of the above case. A foreknowledge of a gastric ulcer or gall stones would encourage a most meticulous scrutiny in the event of later acute abdominal pain.

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COMPLETE INVERSION OF THE PUERPERAL UTERUS*

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REPORT OF CASE

THE patient is a primipara, twenty-six years old. In 1917 she had an operation for swollen glands in left side of neck; in 1921 a "nervous breakdown"; and in 1927 an appendectomy.

Her last regular menstruation was on March 28, 1930. Pregnancy was normal except for nausea and vomiting throughout. Patient was admitted to San Diego County Hospital on November 24, 1930, for vomiting of pregnancy, and was discharged on December 4, 1930, improved. She attended the prenatal clinic at the County Hospital four or five times. Blood pressure, urinary findings and weight normal. Blood Wassermann negative.

Labor began at 6 p. m. December 25, 1930. Patient was admitted to the Obstetrical Service of the San Diego County Hospital at 7:45 p. m. Membranes ruptured spontaneously at 8:30 p. m. At 9:30 p. m. the intern delivered the patient of a live female baby, weighing five pounds five ounces. He admitted using considerable pressure on the fundus and traction on the cord in order to express the placenta. At 9:40 p. m. the placenta attached to the fundus suddenly came down through the lower opening of the birth canal, carrying with it the entire inverted uterus. At no time had pituitrin been administered to the patient.

The intern at once summoned the resident. The latter telephoned me to come to the hospital. On my arrival in the delivery room at 10:15 p. m., the completely inverted uterus with placenta attached to the

* Read at a meeting of the Los Angeles Obstetrical and Gynecological Society, December 8, 1931.